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L2 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2001:95767 CAPLUS
TITLE: New signaling pathways for hormones and cyclic adenosine 3',5'-monophosphate action in endocrine cells
AUTHOR(S): Richards, JoAnne S.
CORPORATE SOURCE: Department of Molecular and Cellular Biology, Baylor College of Medicine, Houston, TX, 77030, USA
SOURCE: Mol. Endocrinol. (2001), 15(2), 209-218
CODEN: MOENEN; ISSN: 0888-8809
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The glycoprotein hormones, ACTH, TSH, FSH, and LH regulate diverse functions in endocrine cells. Although cAMP and PKA have long been shown to mediate specific intracellular signaling events including the transcription of specific genes via the CREB-CBP complex, recent observations have indicated that PKA does not account for all of the intracellular targets of cAMP. For example, TSH stimulation of thyroid cell proliferation is not completely blocked by PKA inhibitors. TSH and FSH can stimulate PKB phosphorylation by a PKA-independent but PI3-K/PDK1-dependent pathway. An FSH inducible kinase, Sgk, has recently been shown to be a close relative of PKB. Sgk is also a target of PI3-K-PDK1 pathway, indicating that some effects previously ascribed to PKB may be mediated by this inducible kinase. The identification of novel cAMP-binding proteins that exhibit guanine nucleotide exchange (GEF) activity (**cAMP-GEFS**; Epacs) has open new doors for cAMP action that include activation of small GTPases such as Rap1a, Rap2, and possibly Ras. These GTPases are known activators of down-stream kinase cascades, including p38MAPK and Erk1/2 as well as PI3-K. Thus, FSH and TSH activation of PKB and Sgk may occur via this alternative cAMP

pathway that involves **cAMP-GEFs** and the activation of the PI3-K/PDK1 pathway.

L2 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:291074 CAPLUS
DOCUMENT NUMBER: 132:318614
TITLE: Mammalian Rap1A and Ras-associated guanine nucleotide exchange proteins and cDNAs and methods for diagnosis, therapy, and drug screening
INVENTOR(S): Kawasaki, Hiroaki; Graybiel, Ann; Housman, David
PATENT ASSIGNEE(S): Massachusetts Institute of Technology, USA
SOURCE: PCT Int. Appl., 128 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2000024768 | A2 | 20000504 | WO 1999-US24826 | 19991022 |
| WO 2000024768 | A3 | 20001109 | | |

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RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1998-105507 19981023
US 1998-108685 19981116

AB The present invention describes the identification, isolation, sequencing and characterization of two human CalDAG-GEF, and two human **cAMP-GEF** genes, which are assocd. with the Ras pathway. Also identified are CalDAG-GEF gene homologs in mice and **cAMP-GEF** gene homologs in rats. Nucleic acids and proteins comprising or derived from the CalDAG-GEFs and/or **cAMP-GEFs** are useful in screening and diagnosing certain Ras-assocd. cancers, in identifying and developing therapeutics for treatment of certain Ras-assocd. cancers, and in producing cell lines and transgenic animals useful as models of Ras-assocd. cancers. Thus, CalDAG-GEFI was found to increase GTP-bound Rap1A and this increase was augmented in the presence of the calcium ionophore A23187 or the phorbol ester TPA. CalDAG-GEFI reduced RasV12 activation of Elk1 by .apprx.4-fold. Northern anal. indicated that human CalDAG-GEFI is expressed strongly in the brain and that the mRNA for this protein is strikingly enriched in the striatum. Further, the CalDAG-GEFI is synthesized in striatal projection neurons and is transported to striatopallidal and striatonigral terminals. CalDAG-GEFII activated Ras, but not Ral1A or Rap1A. Unlike CalDAG-GEFI, CalDAG-GEFII increased the transcriptional activity of Elk1 downstream to Erk/MAP kinase. Northern anal. showed highest expression of CagDAG-GEFII in the cerebellum, cerebral cortex, and amygdala, with low expression in the striatum. CAMP-GEFI and II strongly and selectively activated Rap1A, but not Ras or Ral1A, in the presence of cAMP. Northern anal. indicated that cAMP-GEFI was widely expressed while cAMP-GEFII was expressed selectively in the brain and adrenal glands.

IT cDNA sequences
(for mammalian Rap1A and Ras-assocd. guanine nucleotide exchange proteins CalDAG-GEF and **cAMP-GEF**)

IT Protein sequences
(of mammalian Rap1A and Ras-assocd. guanine nucleotide exchange proteins CalDAG-GEF and **cAMP-GEF**)

L2 ANSWER 3 OF 8 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000414769 MEDLINE
DOCUMENT NUMBER: 20372739
TITLE: Rap2 as a slowly responding molecular switch in the Rap1 signaling cascade.
AUTHOR: Ohba Y; Mochizuki N; Matsuo K; Yamashita S; Nakaya M; Hashimoto Y; Hamaguchi M; Kurata T; Nagashima K; Matsuda M
CORPORATE SOURCE: Department of Pathology, Research Institute, International Medical Center of Japan, Shinjuku-ku, Tokyo 162-8655, Japan.
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (2000 Aug) 20 (16) 6074-83.
Journal code: NGY. ISSN: 0270-7306.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY WEEK: 20001101
AB . . . 50% of total Rap2 protein in adherent cells. Guanine nucleotide exchange factors (GEFs) for Rap1, C3G, Epac (or cyclic AMP [**cAMP**]-**GEF**), CalDAG-GEFI, PDZ-GEF1, and GFR efficiently increased the level of GTP-Rap2 both in 293T cells and in vitro. GTPase-activating proteins (GAPs). . . .

L2 ANSWER 4 OF 8 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000431014 MEDLINE
DOCUMENT NUMBER: 20420809
TITLE: Quantitative determination of Rap 1 activation in cyclic nucleotide-treated HL-60 leukemic cells: lack of Rap 1 activation in variant cells.
AUTHOR: von Lintig F C; Pilz R B; Boss G R
CORPORATE SOURCE: Department of Medicine and Cancer Center, University of California, San Diego, La Jolla, California, CA 92093-0652, USA.
CONTRACT NUMBER: R01GM055586 (NIGMS)
R21 CA81115 (NCI)
SOURCE: ONCOGENE, (2000 Aug 17) 19 (35) 4029-34.
Journal code: ONC. ISSN: 0950-9232.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 200011
ENTRY WEEK: 20001103
AB . . . parental cells. Thus, cAMP activation of Rap 1 in HL-60 cells is likely through a cAMP-regulated guanine nucleotide exchange factor ( **cAMP-GEF**) and since cAMP does not activate Rap 1 in the variant cells, the data suggest that full post-translational processing of Rap 1 is necessary for **cAMP-GEF** activation of Rap 1. Activation of Rap 1 by cGMP analogs has not been previously found and suggests possible cross-talk. . .

L2 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:721365 CAPLUS
DOCUMENT NUMBER: 133:317777
TITLE: Follicle-stimulating hormone (FSH) stimulates phosphorylation and activation of protein kinase B (PKB/Akt) and serum and glucocorticoid-induced kinase (Sgk): evidence for A kinase-independent signaling by FSH in granulosa cells
AUTHOR(S): Gonzalez-Robayna, Ignacio J.; Falender, Allison E.; Ochsner, Scott; Firestone, Gary L.; Richards, JoAnne

S.
CORPORATE SOURCE: Department of Molecular and Cellular Biology, Baylor College of Medicine, Houston, TX, 77030, USA
SOURCE: Mol. Endocrinol. (2000), 14(8), 1283-1300
CODEN: MOENEN; ISSN: 0888-8809
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 76
REFERENCE(S): (1) Adams, S; Nature 1991, V349, P694 CAPLUS
(2) Adashi, E; Endocrinology 1988, V122, P1583 CAPLUS
(3) Agati, J; J Biol Chem 1998, V273, P18751 CAPLUS
(4) Alessi, D; EMBO J 1996, V15, P6541 CAPLUS
(5) Alliston, T; Endocrinology 2000, V141, P385
CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB FSH stimulates in ovarian granulosa cells diverse, differentiation-dependent responses that implicate activation of specific cellular signaling cascades. In these studies three kinases were investigated to det. their relationship to FSH, cAMP, and A kinase signaling: protein kinase B (PKB/Akt), serum and glucocorticoid-induced kinase (Sgk), and p38 mitogen-activated protein kinase (p38MAPK). The phosphorylation (activation) of these kinases was analyzed by using selective agonists/inhibitors: forskolin/H 89 for cAMP-dependent protein kinase (A kinase), insulin-like growth factor I (IGF-I)/LY 294002 and wortmannin for phosphatidylinositol-dependent kinase (PI3-K), and phorbol myristate (PMA)/GF 109203X for diacylglycerol and Ca++-dependent kinases (C kinases). An inhibitor (PD 98059) of MEK1, which regulates extracellular regulated kinases (ERKs), and SB 203580, which inhibits p38MAPK, were also used. In addn., we analyzed the expression of the recently described, cAMP-regulated guanine nucleotide exchange factors (cAMP-GEFI and GEFII) that impact Ras-related GTPases and Raf kinases, known regulators of various protein kinase cascades. We provide evidence that FSH, forskolin, and 8-bromo-cAMP stimulate phosphorylation of PKB by mechanisms involving PI3-K (LY 294002/wortmannin sensitive) not A kinase (H 89 insensitive), a pattern of response mimicking that of IGF-I. In contrast, FSH induction and phosphorylation of Sgk protein requires A kinase (H 89 sensitive) but also involves PI3-K (LY 294002 sensitive) as well as p38MAPK (SB 203580 sensitive) pathways. PMA (C kinase) abolished FSH-mediated (but not IGF-I-mediated) phosphorylation of PKB at a step(s) upstream of PI3-K and independent of A kinase. Lastly, FSH-mediated phosphorylation of p38MAPK is neg. affected by A kinase and PI3-K, suggesting that it may be downstream of specific members of the **cAMP-GEF**/Rap/Raf pathway. We propose that cAMP activation of A kinase is obligatory for transcription of Sgk in granulosa cells whereas cAMP (IGF-I-like)-mediated phosphorylation (activation) of PKB and Sgk (via PI3-K), as well as p38MAPK, involves other cellular events. These results provide new and exciting evidence that cAMP acts in granulosa cells by A kinase-dependent and -independent mechanisms, each of which controls specific kinase cascades.

L2 ANSWER 6 OF 8 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 3
ACCESSION NUMBER: 2001:27587 BIOSIS
DOCUMENT NUMBER: PREV200100027587
TITLE: Expression of cAMP-regulated guanine nucleotide exchange factors in pancreatic beta-cells.
AUTHOR(S): Leech, Colin A.; Holz, George G.; Chepurny, Oleg; Habener, Joel F. (1)
CORPORATE SOURCE: (1) Laboratory of Molecular Endocrinology, Massachusetts General Hospital, 55 Fruit Street, WEL320, Boston, MA, 02114: jhabener@partners.org USA

SOURCE: Biochemical and Biophysical Research Communications, (November 11, 2000) Vol. 278, No. 1, pp. 44-47. print. ISSN: 0006-291X.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB. . . remains unknown. Here we present evidence for the expression of type
1 and type 2 cAMP-regulated guanine nucleotide exchange factors ( **cAMP-GEFs**) in beta-cells. GEFs are activated by their binding of cAMP. Because **cAMP-GEFs** activate Ras/MAPK proliferation signaling pathways, they may play an important role in PKA-independent, GLP-1-mediated, signaling pathways in the regulation of. . .

L2 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:136940 CAPLUS
DOCUMENT NUMBER: 130:178854
TITLE: Isolation and characterization of a novel family of guanine nucleotide exchange factors (GEF) directly regulated by second messenger systems. Novel second messenger pathways
AUTHOR(S): Kawasaki, Hiroaki
CORPORATE SOURCE: Cent. Cancer Res., Massachusetts Inst. Technol., USA
SOURCE: Jikken Igaku (1999), 17(4), 490-494
CODEN: JIIGEF; ISSN: 0288-5514
PUBLISHER: Yodosha
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review with 12 refs., on isolation, distribution, and the structure of a
novel family of guanine nucleotide exchange factors (GEF) having second messenger-binding sites, i.e. **cAMP-GEF** (**cAMP**
-regulated GEF) and CalDAG-GEF (calcium and diacylglycerol-regulated GEF),
regulation of **cAMP-GEF** and CalDAG-GEF by second messenger mols., their substrate specificity, and substrate-specific activation of Rap1 by second messenger mols. through activation of GEF domain.

L2 ANSWER 8 OF 8 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999074384 MEDLINE
DOCUMENT NUMBER: 99074384
TITLE: A family of cAMP-binding proteins that directly activate Rap1.
AUTHOR: Kawasaki H; Springett G M; Mochizuki N; Toki S; Nakaya M; Matsuda M; Housman D E; Graybiel A M
CORPORATE SOURCE: Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology (MIT), Cambridge, MA, 02139, USA.
CONTRACT NUMBER: R01 HD28341 (NICHD)
P01 CA42063 (NCI)
P01 HL41484 (NHLBI)
+
SOURCE: SCIENCE, (1998 Dec 18) 282 (5397) 2275-9.
Journal code: UJ7. ISSN: 0036-8075.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
OTHER SOURCE: GENBANK-U78167; GENBANK-U78168; GENBANK-U78516; GENBANK-U78517
ENTRY MONTH: 199903
AB . . . brain and body organs and that exhibit both cAMP-binding and guanine nucleotide exchange factor (GEF) domains is reported. These cAMP-regulated **GEFs** (**cAMP-GEFs**) bind cAMP and selectively activate the Ras superfamily guanine nucleotide binding

protein Rap1A in a cAMP-dependent but PKA-independent manner. Our. . .

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